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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/577,084	05/24/2000	Keiya Ozawa	50026/012002	5150

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EXAMINER	
HOWARD, ZACHARY C	
ART UNIT	**PAPER NUMBER**
1646	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/577,084	OZAWA ET AL.	
	Examiner	Art Unit	
	Zachary C. Howard	1646	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *27 February 2007*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,4-8,10,14,15,18,20 and 22-26* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,4-8,10,14,15,18,20 and 22-26* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *25 May 2000* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *1/4/07; 4/9/07*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Status of Application, Amendments and/or Claims

The amendment of 2/27/07 has been entered in full. Claims 1 and 10 are amended. It is noted that the first sentence of page 6 of Applicants' 2/27/07 response states that "[c]laims 1, 4-8, 10, 14, 15, 17, 18, 20, and 22-26 are pending". However, claim 17 was cancelled in Applicants' 6/2/06 response (see pg 7, line 2 and also pg 4 of the claim listing). The claim listing (titled "Amendments to the Claims") accompanying Applicants' 2/27/06 response correctly indicates that claim 17 is canceled (see pg 4).

Claims 1, 4-8, 10, 14, 15, 18, 20 and 22-26 are pending and under consideration.

Information Disclosure Statement

The Information Disclosure Statements of 1/4/07 and 4/9/07 have been considered.

Withdrawn Objections and/or Rejections

The following page numbers refer to the previous Office Action (8/23/06).

The rejection of claim 25 under 35 U.S.C. § 112, first paragraph at pg 3-5 for containing new matter is *withdrawn in part* in view of Applicants' persuasive arguments at pg 9-10 of the 2/27/07 response. Claim 25 was rejected for containing new matter because the claim is directed to a kit comprising two vectors (the vector of claim 7 or claim 10 and a vector comprising an exogenous gene) and a ligand, but the specification did not appear to describe said kit. Applicants' response at pg 9-10 points to teachings in the specification that describe said kit. Therefore, this basis for the new matter rejection is withdrawn. However, please note that claim 25 remains rejected for encompassing new matter for the reasons set forth below.

The rejection of claims 1, 4-8, 10, 14, 15, 18, 20 and 22-26 under 35 U.S.C § 112, second paragraph, at pg 5 for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention is *withdrawn* in view of Applicants' amendments to the claims.

Maintained Objections and/or Rejections

Claim Rejections - 35 USC § 112, 1st paragraph

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1, 4-8, 20, 22, 23, 25 and 26 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement because the claims contain new matter. This rejection was set forth at pg 3-4 of the 8/23/06 Office Action. For clarity, the rejection set forth previously is first repeated, and then Applicants' arguments concerning this rejection are addressed.

Claim 1 was amended 6/2/06 to include the limitation that the "second polypeptide comprises the extracellular region of a granulocyte colony stimulating factor receptor and the cytoplasmic region of c-mpl". It is recognized that this amendment incorporates a limitation that was previously present in dependent claim 22 (which was a new claim submitted 5/4/04). However, in view of the specification as originally filed on 5/20/00 this limitation constitutes new matter.

In making the amendment to claim 1, Applicants' 6/2/06 response (pg 6) points to support in the specification at page 31, lines 19-24. However, this portion of the specification refers to the "[t]he extracellular region of ΔGCR and the cytoplasmic domain of c-mpl". Therefore, while the instant claim refers to the "the extracellular region" of granulocyte colony stimulating factor receptor (also known as G-CSFR or GCR), the specification refers to the "the extracellular region of ΔGCR". These two phrases differ in scope. The extracellular region of G-CSFR encompasses the entire extracellular region of the full-length receptor, while the ΔGCR molecule "is deficient in the 5th residue, Glu, through the 195th residue, Leu, of the G-CSF receptor extracellular domain" (see pg 14, lines 3-4). Furthermore, the specification only teaches working examples of fusion proteins comprising ΔGCR and c-mpl (e.g., Figure 20). There is no teaching in the specification describing a receptor comprising the full-length G-CSFR

extracellular domain and the cytoplasmic domain of c-mpl. Therefore, there is no conception of this specific genus of molecules in the specification, nor does the concept of the specific genus flow naturally from the disclosure. As such, the specification as originally filed lacks support for the genus of molecules encompassed by the amended claims. Claims 4-8, 20, 23, 25 and 26 depend from claim 1 and therefore encompass new matter for the same reason. Claim 22 depends from claim 10 but includes a further limitation of similar nature to claim 1; that is, claim 22 limits the claimed vector to encoding a second polypeptide comprising the extracellular region of G-CSFR and c-mpl. Therefore, claim 22 encompasses new matter for the same reason as claim 1.

Applicants' arguments (2/27/07; pg 7-9) as they pertain to the rejection have been fully considered but are not deemed to be persuasive for the following reasons.

In the response, Applicants argue that "in view of the various examples in Applicants' specification of fusion proteins comprising the full-length G-CSF receptor extracellular portion and fusion proteins containing the c-mpl cytoplasmic domain, Applicants submit that one skilled in the art would recognize that other combinations of the disclosed domains, including a fusion protein containing the full-length G-CSF receptor extracellular domain and the c-mpl cytoplasmic domain are within the scope of the invention described in the specification as filed" (pg 8-9). Applicants point to statements in the specification on pg 4, lines 13-16; pg 7, lines 18-22; and pg 5, lines 1-4; Figure 14 (parts A and D); and Example 8 in support.

Applicants' arguments have been fully considered but are not found persuasive. The various examples in the specification cited by Applicants do not provide support for the written description of a fusion protein comprising the full-length extracellular domain of G-CSFR (as opposed to the extracellular region of ΔGCR) and c-mpl. A genus may not support a subgenus even though there is a disclosed species within the subgenus (*In re Smith*. 173 USPQ 679 (CCPA 1972)). Applicants point to a teaching in the specification regarding a genus of fusion proteins; specifically the statement at pg 4, lines 13-16, which is directed to a genus of fusion proteins comprising "a ligand-binding domain", a "domain that associates when a ligand binds to the ligand binding domain"; and a "domain that imparts proliferation activity to a cell upon association". Applicants

also point to a teaching in the specification regarding a species within this genus; specifically, Example 8 which describes a fusion protein (ΔGCR-Mpl-TmR) containing a specific ligand-binding domain (TmR); a specific domain that associates when a ligand binds to the ligand binding domain (ΔGCR); and a domain that imparts proliferation activity to a cell upon association (Mpl). However, the claims are directed to fusion proteins "comprising the extracellular region of a granulocyte colony stimulating factor", which encompasses the full-length G-CSFR receptor. The disclosure of the single species of extracellular domain with a deletion (ΔGCR) does not provide support for the subgenus encompassing the "extracellular region" of G-CSF, which includes the full-length extracellular domain without a deletion.

Applicants also point to teachings in the specification regarding the "domain which imparts proliferation activity to a cell"; specifically, statements at pg 5 and 7. The statement at pg 7, lines 18-22, teaches that this domain can be "an entire molecule of a cytokine receptor" or "only a domain in a molecule that imparts proliferating activity to a cell". This teachings fails to provide written description for a fusion protein comprising the full-length extracellular domain of G-CSFR and c-mpl, because the full-length extracellular domain is neither "an entire molecule of a cytokine receptor" or a domain with proliferating activity. The specification does not teach that the extracellular domain of G-CSFR is an example of a domain with proliferating activity. The statement at pg 5, lines 1-4 teaches, that the proliferating domain "may be derived from a G-CSF receptor or c-mpl". Again, this teaching fails to provide written description for a fusion protein comprising the full-length extracellular domain of G-CSFR and c-mpl, because the specification does not teach that the extracellular domain of G-CSFR is an example of a domain with proliferating activity.

Applicants also point to Figure 14 for support for written description for the claims. However, fails to provide written description for a fusion protein comprising the full-length extracellular domain of G-CSFR and c-mpl, because Figure 14 teaches different species of protein (e.g., a full-length extracellular domain of the G-CSF receptor (part A) or a fusion protein containing the full-length extracellular domain of the G-CSF receptor and a mutant estrogen receptor specific for 4-hydroxytamoxifen (part

D)) but does not provide support for the particular claimed subgenus. Furthermore, there is no teaching in the specification that specifically suggest combining the full-length extracellular domain from these particular proteins (as opposed to ΔGCR) with a c-mpl proliferating domain.

In *Purdue Pharma L.P. v. Faulding Inc.*, 230 F.3d 1320, 1326, 56 USPQ2d 1481, 1486 (Fed Cir. 2000), the court noted that with respect to *In re Ruschig* 379 F.2d 990, 154 USPQ 118 (CCPA 1967) that "*Ruschig* makes clear that one cannot disclose a forest in the original application, and then later pick a tree out of the forest and say "here is my invention". In order to satisfy the written description requirement, the blaze marks directing the skilled artisan to that tree must be in the originally filed disclosure".

The Examiner notes Applicants' arguments (pg 10) with regard to support for the genus of "tamoxifen derivatives and metabolites", which was discussed at during the 11/14/06 interview. Applicants' arguments have been fully considered and are found to be persuasive. As pointed out by Applicants, the specification teaches, "tamoxifen, the derivative thereof (ex. Tremifen), or the metabolite thereof (ex. 4-hydroxytamoxifen) can be preferably used" (pg 7). Also as pointed out by Applicants, a number of derivatives and metabolites of tamoxifen were well known in the art at the time of filing, as evidenced by the publications of Gulino et al (1982) and Lien et al (1989). It is noted that this issue was discussed at the 11/14/06 interview but was not the basis of any rejection of record.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 10, 14, 15, 18 and 20 are rejected under 35 U.S.C. 103(a) as being unpatentable over Ito et al, 1997 (Blood. 90(10): 3884-3892; cited previously) in view of Drachman et al (1997. Proc Natl Acad Sci USA. 94: 2350-2355). This rejection was set forth previously and maintained at pg 5-7 of the 8/23/06 Office Action. For clarity, the rejection set forth previously is first repeated, and then Applicants' arguments concerning this rejection are addressed.

Ito teaches a fusion protein comprising the granulocyte colony stimulating factor receptor (G-CSFR) and the estrogen receptor hormone-binding domain (ER-HBD; Fig 1a and pg 3886) that is functional in proliferation of the murine pro-B cell line Ba/F3 (Fig 3). Ito teaches DNA, vectors and isolated cells encoding the fusion protein (pg 3885). Ito teaches a mutant ER protein that binds synthetic 4-hydroxytamoxifen but not estrogen and teaches the advantage of using this mutant ER in the fusion protein of the invention (pg 3891). Ito further teaches co-transfection with a plasmid comprising the exogenous blasticidin gene. Therefore, Ito teaches all of the limitations of the claims, except that (1) the vector encodes a fusion protein comprising c-mpl and (2) the exogenous gene and the DNA encoding the fusion protein are present on the same molecule (i.e., a single vector rather than two discrete vectors). Ito further teaches that "[t]he strategy used in this study is based on the finding that estrogen can activate fusion proteins between ER-HBD and a wide variety of heterologous proteins" (pg 3888) and "[v]arious modifications are possible to improve the system as described here. We are also constructing similar chimeric genes using other growth factor receptor genes such as *c-kit* and erythropoietin receptor" (pg 3891).

Drachman teaches a vector encoding full-length c-mpl and also comprising an exogenous neomycin resistance gene (See "Receptor Constructs, pg 2351). Drachman teaches that this vector encodes a c-mpl protein that supports proliferation of Ba/F3 cells in response to thrombopoietin (Fig 2). Drachman further teaches that "[l]ike the receptors for growth hormone, erythropoietin and granulocyte colony-stimulating factor, Mpl is believed to be activated through homodimerization..." (pg 2350).

It would have been obvious to the person of ordinary skill in the art at the time the invention was made to substitute c-mpl as taught by Drachman for G-CSFR in the

vector taught by Ito, and to further include an exogenous gene encoding neomycin resistance as taught by Drachman. The person of ordinary skill in the art would be motivated to do so in order to use the vector to selectively amplify Ba/F3 hematopoietic cells, and because Ito suggests modifications using other growth factor receptor genes such as the erythropoietin receptor. The person of ordinary skill in the art would have had a reasonable expectation of success because Ito teaches all of the techniques necessary to make a vector encoding fusion protein between a receptor and ER-HBD, and to use it for proliferation of Ba/F3 hematopoietic cells, and in the absence of other evidence, c-mpl would work as well as G-CSFR in the fusion protein encoded by the vector.

Applicants' arguments (2/27/07; pg 12-14) as they pertain to the rejection have been fully considered but are not deemed to be persuasive for the following reasons

At pg 12, Applicants argue that Drachman does not teach or suggest generating a vector encompassed by claim 10. Applicants state the Drachman describes vectors containing functional elements of the Mpl cytoplasmic domain and a neomycin gene, but fails to describe a vector encoding a fusion protein or a vector encoding a fusion protein containing the ligand-binding domain of a steroid hormone receptor and c-mpl. Applicants further state that Ito describes vectors containing a DNA encoding a fusion protein including a G-CSFR and the hormone-binding domain of an estrogen receptor.

Applicants' arguments have been fully considered but are not found persuasive. In this portion of the response, Applicants appear to be individually attacking each of the references (Drachman and Ito) used in the 103(a) rejection. In response to Applicants' arguments against the references individually, one cannot show nonobviousness by attacking references individually where the rejections are based on combinations of references. See *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981); *In re Merck & Co.*, 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986). The 103(a) rejection set forth previously and maintained herein is based on the combination of teaching of the references of Drachman and Ito.

At pg 12-13, Applicants refer to their previous argument (made on 6/2/06) that Ito "teaches away from the desirability of making a vector expressing a fusion protein with a

signal transducing portion other than a portion from G-CSFR". Applicants refer to the PTO's response to this argument which states "Ito teaches other uses for the chimeric receptors than clinical *in vivo* application. Specifically, Ito teaches that the chimeric receptors can be used for *ex vivo* amplification of cells". In response to this, Applicants argue "that the passages referred to by the Office refer to ex vivo amplification prior to *in vivo* use". Applicants point to Ito at page 3884 as suggesting that ex vivo expansion of transduced cells can overcome the problems associated with low transduction efficiency. Applicants argue that the "purpose of the expansion is clearly to produce a sufficient number of transduced cells to obtain a cell population with an expression level required for improvement of clinical manifestations" and that the statements in Ito regarding the safety of signals G-CSFR derived molecules as opposed to other receptors also applies to *ex vivo* amplification.

Applicants' arguments have been fully considered but are not found persuasive. Despite teaching that G-CSFR derived molecules may be the safest for clinical application, Ito clearly also suggests making "similar chimeric genes using other growth factor receptor genes such as *c-kit* and erythropoieitin receptor genes". As noted previously, Ito also teaches that the "modified amplifier gene will be applicable to the *ex vivo* expansion of transduced hematopoietic stem cells". This argument is not found to be persuasive. As shown by Ito in Figure 4, *ex vivo* expanded Ba/F3 cells can also be used for continuous culture over a period of at least 24 days.

Claim 24 is rejected under 35 U.S.C. 103(a) as being unpatentable over Ito et al, 1997 (Blood. 90(10): 3884-3892; cited previously) in view of Drachman et al (1997. Proc Natl Acad Sci USA. 94: 2350-2355) as applied to claim 10 above, and further in view of Picard, 1999 (Picard, Ch 11 (pg 261-274) in Nuclear Receptors: a Practical Approach (D. Picard, ed) Oxford University Press, Oxford, 1999). This rejection was set forth at pg 7-8 of the 8/23/06 Office Action. For clarity, the rejection set forth previously is first repeated, and then Applicants' arguments concerning this rejection are addressed.

The teachings of Ito and Drachman as applied to claim 10 are described above. Neither Ito nor Drachman teaches steroid receptor hormone binding domains from androgen, progesterone, glucocorticoid or mineral corticoid receptor.

Picard teaches "Regulation of heterologous proteins by fusion to a hormone binding domain" (see title, pg 261). Picard further teaches fusions using the hormone-binding domain (HBD) of either the estrogen, androgen, progesterone, glucocorticoid or mineral corticoid receptor (Table 1). Picard teaches that each of these steroid receptors forms a stable complex on ligand binding (pg 268).

It would have been obvious to the person of ordinary skill in the art at the time the invention was made to substitute c-mpl as taught by Drachman for G-CSFR in the vector taught by Ito, and to further include an exogenous gene encoding neomycin resistance as taught by Drachman, and to further substitute any of the steroid hormone receptor ligand binding domains taught by Picard. The person of ordinary skill in the art would be motivated to do so in order to use the vector to selectively amplify Ba/F3 hematopoietic cells, and because Ito suggests modifications using other growth factor receptor genes such as the erythropoietin receptor, and because Picard teaches the interchangeability of steroid hormone receptor ligand binding domains in heterologous fusion proteins. The person of ordinary skill in the art would have had a reasonable expectation of success because Ito teaches all of the techniques necessary to make a vector encoding fusion protein between a receptor and ER-HBD, and to use it for proliferation of Ba/F3 hematopoietic cells, and in the absence of other evidence, c-mpl would work as well as G-CSFR, and the androgen, progesterone, glucocorticoid or mineral corticoid receptor would work as well as the estrogen receptor, in the fusion protein.

Applicants' arguments (2/27/07; pg 14-15) as they pertain to the rejection have been fully considered but are not deemed to be persuasive for the following reasons.

In the response, Applicants argue that dependent claim 24 is non-obvious over Ito in view of Drachmann in further view of Picard for the same reasons that claim 10 is non-obvious over Ito in view of Drachmann.

Applicants' arguments have been fully considered but are not found persuasive. Applicants' arguments regarding claim 10 were addressed above. Applicants' arguments regarding claim 24 are not found to be persuasive for the same reason as the arguments regarding claim 10.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., In re Berg, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); In re Goodman, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); In re Longi, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); In re Van Ornum, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); In re Vogel, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and In re Thorington, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 10, 14, 15, 18, 20 and 24 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claim 8, 12, 15, 17 and 22 of copending Application No. 09/905592 in view of Ito et al, 1997 (Blood. 90(10): 3884-3892; cited previously) and further in view of Drachman et al (1997. Proc Natl Acad Sci USA. 94: 2350-2355). It is noted that the claims of the '592 application were last amended 2/27/07. This provisional rejection was set forth at pg 8-10 of the 8/23/06 Office Action.

The '592 application is a divisional application of copending Application No. 09/142305. As noted in the previous Office Action, the instant application is a CIP of

'305 and claims priority to its filing date. However, the disclosure of '305 does not disclose the fusion proteins comprising c-mpl. Therefore, with regard to the species of c-mpl, the instant application does not merit priority to the filing date of '305.

Claims 8, 12, 15, 17 and 22 of '592 contain all of the limitations of claims 10, 14, 15, 18 and 20 of the instant application, except that the vector is directed to one encoding the G-CSF receptor in the '592 application, and is limited to one encoding c-mpl in the instant application.

The teaching of Ito and Drachman are described above in the section entitled "Claim Rejections - 35 USC § 103".

It would have been obvious to the person of ordinary skill in the art at the time the invention was made to substitute c-mpl as taught by Drachman for G-CSFR in the vector encoding the fusion protein taught by the '592 application. The person of ordinary skill in the art would be motivated to do so in order to use the vector to selectively amplify Ba/F3 hematopoietic cells, and because Ito suggests modifications using other growth factor receptor genes such as the erythropoietin receptor. The person of ordinary skill in the art would have had a reasonable expectation of success because the '592 application teaches all of the techniques necessary to make a vector encoding fusion protein between a receptor and ER-HBD, and to use it for proliferation of Ba/F3 hematopoietic cells, and in the absence of other evidence, c-mpl would work as well as G-CSFR in the fusion protein encoded by the vector.

This is a provisional obviousness-type double patenting rejection.

Applicants' arguments (2/27/07; pg 15) as they pertain to the rejection have been fully considered but are not deemed to be persuasive for the following reasons.

In the response, Applicants "agree to address this basis for rejection, if appropriate, upon an indication of otherwise allowable subject matter".

Applicants' arguments have been fully considered but are not found persuasive. The Examiner notes that Applicants agree to address this provisional basis for rejection upon indication of otherwise allowable subject matter. However, the Examiner notes that, at present, the provisional rejection remains appropriate and therefore the rejection is maintained for the reasons set forth previously and reiterated above.

Conclusion

No claims are allowed.

THIS ACTION IS MADE FINAL. See MPEP § 706.07(a). Applicants are reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Zachary C. Howard whose telephone number is 571-272-2877. The examiner can normally be reached on M-F 9:30 AM - 6:00 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary B. Nickol can be reached on 571-272-0835. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Elizabeth C. Kemmerer

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ELIZABETH KEMMERER
PRIMARY EXAMINER